REMARKS

Claims 1-3, 11-12, 15-19, 24 and 25 were pending in the application. Claims 1, 3, 11, 15, 16, 17, 19 and 25 have been amended. Accordingly, after the amendments presented herein have been entered, claims 1-3, 11-12, 15-19, 24 and 25 will remain pending. For the Examiner's convenience, these claims are presented herein in Appendix A.

Support for the amendments to the claims can be found throughout the specification including the originally filed claims. Specifically, support for the amendments to claims 1, 3, 17, 19 and 25 can be found at, for example, page 3, line 25 through page 4, line 14 of the specification. Support for the amendments to claims 11, 15, and 16 can be found at page 3, line 25 through page 4, line 29 of the specification.

No new matter has been added. Any amendments to the claims should in no way be construed as an acquiescence to any of the Examiner's rejections and were done solely to expedite the prosecution of the application. Applicants reserve the right to pursue the claims as originally filed in this or a separate application(s).

Rejection of Claims 1-3, 12, 16-19, and 24-25 Under 35 U.S.C. § 112, First Paragraph

The Examiner has rejected claims 1-3, 12, 16-19, and 24-25 under 35 U.S.C. 112, first paragraph because, according to the Examiner, "***the specification, while being enabling for a method for identifying a compound which binds to PCIP 9q, and thus modulates Kv4.2***, does not reasonably provide enablement for a method for identifying a compound which is suitable for treating any and all cardiovascular disorders." (***Emphasis added***).

While in no way conceding the validity of the Examiner's rejection and solely in the interest of expediting prosecution, Applicants have amended the claims thereby rendering the foregoing rejection moot. Specifically, Applicants have amended the claims to be directed to methods for identifying a compound that binds to and/or modulates the activity of a Kv4.2 or Kv4.3 potassium channel, as suggested by the Examiner. In view of the foregoing, Applicants respectfully request that the Examiner reconsider and withdraw the foregoing rejection.

Rejection of Claims 3, 17, 19, and 24 Under 35 U.S.C. § 112, Second Paragraph

The Examiner has rejected claims 3, 17, 19 and 24 under 35 U.S.C. § 112, second paragraph as being, "indefinite for failing to particularly point out and distinctly claim the

subject matter which applicant regards as the invention." In particular, the Examiner is of the opinion that

> [c]laims 17, 19, and 24 are vague and indefinite because while claims 17 and 19 recite that an EF domain consists of residues 68-252 of human 9Q, claim 24 sets forth ca. 32 short regions in the polypeptide which are defined as EF domains. It is not clear whether the EF domain is the long domain set forth in claim 17 and 19, or only the short regions set forth in claim 24.

The Examiner has further rejected claim 24 under 35 U.S.C. § 112, second paragraph because, according to the Examiner, it does not properly further limit claims 17 and 19.

Applicants respectfully traverse these rejections for the following reasons. Claims 17 and 19 are directed to methods for identifying a compound that binds to and/or modulates the activity of a Kv4.2 or Kv4.3 potassium channel. These methods include contacting a biologically active fragment of a 9q PCIP polypeptide with a test compound. The biologically active fragment can be selected from a number of fragments listed in the Markush group. As recited in the claims, the Markush group consists of (1) an EF domain, (2) a fragment comprising residues 68-252 of human 9q, and (3) an α subunit binding domain from a Kv4.2 or Kv4.3 potassium channel. Therefore, the claims do not provide that the EF domain is comprised of residues 68-252 of the 9q polypeptide. Rather, the claims clearly recite that there are three distinct biologically active fragments of the 9q polypeptide that may be used in the claimed methods. Accordingly, the fragments listed in claim 24 do not render claims 17 and 19 vague and indefinite, but further define the EF domain recited in claims 17 and 19.

In view of the foregoing, Applicants respectfully request that the Examiner reconsider and withdraw the foregoing rejections.

Finally, the Examiner has rejected claims 3 and 19 under 35 U.S.C. § 112, second paragraph as being, "indefinite in the recitation of the term 'interaction'." The Examiner is of the opinion that, "[i]t is not clear whether this term limits the method to finding a candidate compound which will modulate the binding of PCIP 9q to the K channel, or whether the method is directed to finding a candidate compound which will modulate some other functions that the proteins may have."

Applicants respectfully traverse this rejection on the grounds that the claims are clear and definite. Step (b) in claims 3 and 19 requires determining whether the test compound

modulates the interaction of the potassium channel or fragment thereof with the 9q PCIP polypeptide. The term "interaction" is well known in the art and also defined in the Webster's Dictionary to mean "mutual or reciprocal action or influence." Miriam Webster's Collegiate Dictionary, 10th Edition, 1993 Miriam-Webster Incorporated. Thus, claims 3 and 19 encompass determining whether the test compound modulates the reciprocal action or influence of the potassium channel or fragment thereof and the 9q PCIP polypeptide.

Furthermore, as indicated by copies of the issued claims in United States Patents 6,599,875, 6,589,747, and 6,586,259 (attached hereto as Appendices B,C, and D, respectively), the term "interaction" was well known and understood in the art at the time of the invention. Moreover, the presence of the term "interaction" in multiple issued claims[1], demonstrates that this term has been found by the United States Patent and Trademark Office to be clear and definite.

In particular, Applicants would like to respectfully direct the Examiner's attention to U.S. Patent No. 6,436,654 (submitted herewith as Appendix E). This patent was examined by the Examiner (Joseph Murphy) and contains claims directed to methods for identifying compounds which modulate the function of a functional domain of human HIF-1α wherein the modified human HIF-1α exhibits an activity selected from the group consisting of, among others, ***interaction*** with transcriptional coactivator TIF2 in vivo, ***interaction*** with transcriptional coactivator SRC-1 in vivo, and hypoxia-dependent physical ***interaction*** with Ref-1 (see claim 45; *Emphasis added*). A review of the patent specification indicates that the patentees did not define the term "interaction" in the specification. Thus, the Examiner must have found the term "interaction" to be clear and definite based on the fact that this term is well known in the art. Similarly, in the present case the term "interaction" is well known and understood in the art.

In view of all of the foregoing, it is Applicants' position that the term "interaction," as used in the pending claims, is clear and definite and would be understood as such by the ordinarily skilled artisan. Therefore, Applicants respectfully request that the Examiner reconsider and withdraw the foregoing rejection.

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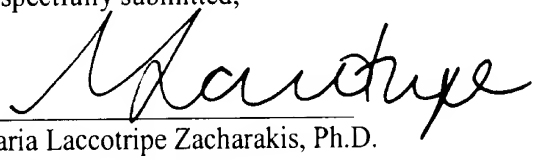
[1] A search of the claims of United States Patents issued since 1976 reveals that the term "interaction" appears in 12,353 patents.

CONCLUSION

Reconsideration and allowance of all the pending claims is respectfully requested. If a telephone conversation with Applicants' Attorney would expedite prosecution of the above-identified application, the Examiner is urged to call the undersigned at (617) 227-7400.

Respectfully submitted,



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Limited Recognition under 37 C.F.R. § 10.9(b)

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Dated: August 20, 2003

APPENDIX A

1. (Currently Amended) A method for identifying a compound that binds to and/or modulates the activity of a Kv4.2 or Kv4.3 potassium channel comprising:

a) contacting a 9q PCIP polypeptide comprising an amino acid sequence selected from the group consisting of SEQ ID NOs: 14, 16, 18, 20, 22, 24, 26, and 28, or a cell expressing said 9q PCIP polypeptide with a test compound; and

b) determining whether said test compound binds to and/or modulates the activity of said 9q PCIP polypeptide, thereby identifying a compound that binds to and/or modulates the activity of a Kv4.2 or Kv4.3 potassium channel.

2. (Previously Presented) The method of claim 1, wherein the binding of said test compound to said 9q PCIP polypeptide, is detected by a method selected from the group consisting of:

a) detection of binding by direct detection of test compound/polypeptide binding;

b) detection of binding using a competition binding assay; and

c) detection of binding using an assay for PCIP activity.

3. (Currently Amended) A method for identifying a compound that binds to and/or modulates the activity of a Kv4.2 or Kv4.3 potassium channel, comprising:

a) incubating a cell expressing i) a potassium channel comprising a Kv4.3 or Kv4.2 subunit, or a fragment thereof that functions as a potassium channel and ii) a 9q PCIP polypeptide comprising an amino acid sequence selected from the group consisting of SEQ ID NOs: 14, 16, 18, 20, 22, 24, 26, and 28, in the presence and absence of a test compound; and

b) determining whether the test compound modulates the interaction of the potassium channel or fragment thereof with said 9q PCIP polypeptide, thereby identifying a compound that binds to and/or modulates the activity of a Kv4.2 or Kv4.3 potassium channel.

11. (Currently Amended) The method of any one of claims 1, 3, 17 or 19 wherein said compound is useful in treating a subject suffering from a cardiovascular disorder associated with an abnormal I_{to} current.

12. (Previously Presented) The method of any one of claims 1, 3, 17 or 19, wherein said 9q PCIP is a human 9q.

15. (Currently Amended) The method of any one of claims 1, 3, 17 or 19, wherein said compound is useful in treating a subject suffering from long-QT syndrome.

16. (Currently Amended) The method of any one of claims 1, 3, 17 or 19, wherein said compound is useful in treating a subject suffering from congestive heart failure.

17. (Currently Amended) A method for identifying a compound that binds to and/or modulates the activity of a Kv4.2 or Kv4.3 potassium channel, comprising:

a) contacting a biologically active fragment of a 9q PCIP polypeptide comprising an amino acid sequence selected from the group consisting of SEQ ID NOs: 14, 16, 18, 20, 22, 24, 26, and 28, wherein said biologically active fragment is selected from the group consisting of an EF domain, residues 68-252 of human 9q, and a Kv4.3 or Kv4.2 potassium channel α subunit binding domain, or a cell expressing said biologically active fragment of said 9q PCIP polypeptide with a test compound; and

b) determining whether said test compound binds to and/or modulates the activity of said biologically active fragment, thereby identifying a compound that binds to and/or modulates the activity of a Kv4.2 or Kv4.3 potassium channel.

18. (Previously Presented) The method of claim 17, wherein the binding of said test compound to said biologically active fragment of said 9q PCIP polypeptide, is detected by a method selected from the group consisting of:

a) detection of binding by direct detection of test compound/biologically active fragment binding;

b) detection of binding using a competition binding assay; and

c) detection of binding using an assay for PCIP activity.

19. (Currently Amended) A method for identifying a compound that binds to and/or modulates the activity of a Kv4.2 or Kv4.3 potassium channel, comprising:

a) incubating a cell expressing i) a potassium channel comprising a Kv4.3 or Kv4.2 subunit, or a fragment thereof that functions as a potassium channel, and ii) a biologically active fragment of a 9q PCIP polypeptide comprising an amino acid sequence selected from the group

consisting of SEQ ID NOs: 14, 16, 18, 20, 22, 24, 26, and 28, wherein said biologically active fragment is selected from the group consisting of an EF domain, residues 68-252 of human 9q, and a Kv4.3 or Kv4.2 potassium channel α subunit binding domain, in the presence and absence of a test compound; and

b) determining whether the test compound modulates the interaction of the potassium channel or fragment thereof with said biologically active fragment of said 9q PCIP polypeptide, thereby identifying a compound that binds to and/or modulates the activity of a Kv4.2 or Kv4.3 potassium channel.

24. (Previously Presented) The method of claim 17 or 19, wherein the EF domain is selected from the group consisting of:

a) residues 116-127, 153-164, 189-200, or 237-248 of SEQ ID NO:14;

b) residues 103-114, 140-151, 176-187, or 224-235 of SEQ ID NO:16;

c) residues 116-127, 153-164, 189-200, or 237-248 of SEQ ID NO:18;

d) residues 98-109, 135-146, 171-182, or 219-230 of SEQ ID NO:20;

e) residues 98-109, 135-146, 171-182, or 219-230 of SEQ ID NO:22;

f) residues 116-127, 103-114, 139-150, or 187-198 of SEQ ID NO:24;

g) residues 66-77, 103-114, 189-200 or 237-248 of SEQ ID NO:26; and

h) residues 98-109, 135-146, 171-182, or 219-230 of SEQ ID NO:28.

25. (Currently Amended) A method for identifying a compound that binds to and/or modulates the activity of a Kv4.2 or Kv4.3 potassium channel comprising:

a) contacting a polypeptide that is at least 95% identical to a 9q PCIP polypeptide comprising an amino acid sequence selected from the group consisting of SEQ ID NOs: 14, 16, 18, 20, 22, 24, 26, and 28 and retains the ability to bind to a Kv4 channel, or a cell expressing said polypeptide with a test compound; and

b) determining whether said test compound binds to and/or modulates the activity of said polypeptide, thereby identifying a compound that binds to and/or modulates the activity of a Kv4.2 or Kv4.3 potassium channel.

-continued

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What is claimed is:

1. An I1-6 antagonist peptide, isolatable from a peptide library by binding tothe intracellular domain of gp130 in a two hybrid system for detecting protein-protein interaction, said peptide comprising SEQ ID NO:2, as well as salts, functional derivatives, and conservatively substituted analogs thereof having IL-6 antagonist activity.

2. The peptide according to claim **1**, comprising the amino acid sequence of SEQ ID NO: 2.

3. The peptide according to claim **1**, consisting of the amino acid sequence of SEQ ID NO:2, as well as salts, functional derivatives, and conservatively substituted analogs thereof having IL-6 antagonist activity.

4. A pharmaceutical composition for prophylaxis, therapy or diagnosis of a hematological disease requiring IL-6 inhibition, comprising the peptide of claim **1** and a pharmaceutically acceptable carrier or excipient.

5. The pharmaceutical composition according to claim **4**, wherein the hematological disease is selected from the group consisting of chronic lymphocytic leukemia (CLL), plasmacytoma/multiple myeloma, Castleman's disease (CD), and anemia.

6. A composition, comprising the peptide of claim **1** and a pharmaceutically-acceptable carrier or excipient.

7. The peptide of claim **1**, wherein said conservatively substituted analogs consist of one or two conservatively substituted amino acid residues in SEQ ID NO:2

8. The peptide according to claim **3**, wherein said conservatively substituted analogs consist of one or two conservatively substituted amino acid residues in SEQ ID NO:2.

* * * * *

All of the references cited herein are hereby incorporated in their entireties by reference.

While this invention has been described with an emphasis upon a preferred embodiment, it will be obvious to those of ordinary skill in the art that variations in the preferred composition and method may be used and that it is intended that the invention may be practiced otherwise than as specifically described herein. Accordingly, this invention includes all modifications encompassed within the spirit and scope of the invention as defined by the following claims.

What is claimed is:

1. A method for identifying compounds that modulate the interaction of amyloid β or its aggregates with a voltage-gated sodium channel, said method comprising:

(a) obtaining a cell that comprises said voltage-gated sodium channel;

(b) contacting said sodium channel with amyloid β or its aggregates in the presence and absence of a test compound; and

(c) determining the activity of said sodium channel in the presence of said test compound as compared with in the absence by measuring the voltage-gated fast inward sodium current within from about 0 to about 30 minutes after said contacting, wherein a test compound that impacts activity is considered a modulator of the interaction of amyloid β or its aggregates with said sodium channel.

2. The method of claim **1**, wherein said cell is a neuronal cell.

3. The method of claim **1**, wherein said cell is a non-neuronal cell.

* * * * *

subjected to mechanical stress such as repeated centrifugation (800×g, 10 minutes), or shearing by forcing the blood through a 21 gauge needle several times prior to testing. In contrast, testing with uncoated microspheres showed no decrease in leukocyte counts. Phase contrast microscopy confirmed leukocyte/platelet complex association with the von Willebrand factor coated microspheres in aggregates and no association with the uncoated microspheres. The aggregates formed by the leukocytes, platelets and von Willebrand factor coated microspheres were noted to be too large to be counted as leukocytes, by the differential cell counter.

Example 3

Study subjects (n=3) destined for Coronary Artery Bypass Graft (CABG) surgery were tested prior to, and throughout the surgical procedure, using the method of the present invention. A differential cell count was performed on citrated whole blood at each time point (pre-surgery, on bypass, post-protamine, intensive care unit, 24 hours post-surgery) throughout the surgical procedure. In addition, 100 μl of citrated whole blood from each time point was added to microwells containing 5 μl (approximately 5×105, 4.5 μm diameter) von Willebrand factor coated microspheres. The microwells were shaken on a rotary shaker at 500 rpm for 1 to 10 minutes A second differential cell count was performed on the blood from the microwells. Taking into account the dilution effect of the bead reagent, the decrease in the number of leukocytes was determined by calculating the ratio between the post reaction counts and the pre-reaction counts and multiplying the result by 100 to obtain the percent decrease in leukocyte count. All study subjects showed loss of leukocytes in the pre-surgical sample as determined by the differential cell counting technique. Microscopy confirmed platelet/leukocyte complex formation on the surface of the VWF coated microspheres. Platelet/leukocyte complex formation in the presence of the VWF coated microspheres was noted in the blood samples collected while the patients were on bypass, however platelet/leukocyte complex formation in the presence of the VWF coated microspheres was not seen in blood samples collected 24 hours post-surgery, in any of the study subjects.

Example 4

Whole blood from a healthy volunteer was drawn into a citrate vacutainer tube, an EDTA vacutainer tube and a heparin vacutainer tube. A drop of blood from each tube was added to the reaction well of three separate reaction cards containing a mixture of VWF-coated polystyrene beads and VWF-coated paramagnetic iron oxide particles (VWF-PIOP) and the suspension mixed vigorously for 5 minutes. Aliquots of whole blood (5 μl) were removed from each card for microscopic wet-mount observation (phase contrast) and stained smear (Hema-3 stain, Fisher Scientific) evaluation. Weak platelet adhesion to the VWF beads was noted with the EDTA blood, with the vast majority of platelets left unbound. Platelets did not associate with VWF-PIOP in the EDTA blood. Both the citrate blood and the heparin blood samples showed extensive binding of platelets to VWF beads with subsequent binding of VWF-PIOP to form large complexes. Few platelets were left unbound. Binding of leukocytes to (or within) these large VWF bead/platelet/VWF-PIOP complexes was not observed either by wet mount or stained smear. Leukocytes and platelets in complex were observed to be associated with the von Willebrand factor coated microspheres and VWF-PIOP in the citrate and heparin samples only when the platelets displayed hyperactivity or when the platelets and/or leukocytes were subjected to mechanical stress such as repeated centrifugation (800×g, 10 minutes). Mechanical stress did not augment platelet association with the VWF Beads or VWF-PIOP, nor did it promote leukocyte/platelet complex formation in the EDTA blood sample.

Preparation of test card—Into a test card, such as that in Oberhardt, U.S. Pat. No. 5,110,727, having a reaction chamber of approximately 30 μl, is placed a reagent composition containing the above noted magnetic and non-magnetic particles coated with vWf, in amounts such that the reagent composition comprises 1–2 mg of coated PIOP particles per ml and from 2×106 to 8×106 polystyrene particles per ml. Also placed into the reaction chamber is an amount of a leukocyte marker, such as FITC-labeled anti-CD45, sufficient to provide a detectable signal. Once the reaction chamber is filled, the sample is then frozen and lyophilized, as described for the preparation of test cards in the above noted Oberhardt patents.

However, the ratio of coated magnetic particles to coated non-magnetic particles is not limited and can be any ratio so long as there are sufficient magnetic particles to form the rotating ring and collapse to the disc or dot.

Platelet/Leukocyte Interaction Test. A disposable or test card containing the reagents described above is placed on a platform above a rotating magnet. Whole blood (or other blood-derived) sample is added to a well, which is pulled by capillary action into the reaction chamber. At that time the magnetic particles and non-magnetic particles are freed, with the magnetic particles forming a rotating ring around the central portion of the reaction chamber. As the reaction progressed, the inner edge of the rotating ring migrated toward the center, with the final endpoint providing a full collapse of the inner edge to the central point to form a disc or dot. The total time elapsed is approximately 1 to 20 minutes, typically in the 2–4 minute range.

When the above noted vWf coated PIOP and vWf coated polystyrene particles are used, the presence of platelet/leukocyte interaction is determined by detection of a leukocyte marker present in the original reagent formulation. Alternatively, if the PIOP are coated with a leukocyte ligand (instead of vWf) the occurrence of the platelet/leukocyte interaction is detected by collapse of the PIOP ring itself.

Obviously, numerous modifications and variations of the present invention are possible in light of the above teachings. It is therefore to be understood that within the scope of the appended claims, the invention may be practiced otherwise than as specifically described herein.

The present application is based on U.S. Provisional Application No 60/165,462, filed Nov. 15, 1999, the entire contents of which are hereby incorporated by reference.

What is claimed is:

1. A platelet/leukocyte interaction assay method, comprising:

contacting a whole blood or blood-derived sample containing platelets and leukocytes with a platelet/leukocyte interaction assay reagent in the presence of an oscillating or rotating magnetic field, said platelet/leukocyte interaction assay reagent comprising a leukocyte marker compound and a mixture of magnetic and non-magnetic particles, wherein said magnetic particles have bound to an outer surface thereof an amount of a first ligand having an affinity for direct interaction with blood platelets and wherein said non-magnetic particles have bound to an outer surface thereof an amount of a second ligand having an affinity

for direct interaction with blood platelets, wherein said first ligand and said second ligand can be the same or different; and

monitoring movement of the magnetic particles in response to the oscillating or rotating magnetic field, to determine the presence or absence of platelet/leukocyte interaction function, a level of platelet/leukocyte interaction, or both, in the whole blood or blood-derived sample containing platelets and leukocytes as determined by comparing a level of leukocyte marker compound in an aggregate formed of said magnetic and non-magnetic particles to a level of leukocyte marker compound outside of said aggregate.

2. The method of claim **1**, wherein said sample is whole blood.

3. The method of claim **1**, wherein said first ligand is a member selected from the group consisting of von Willebrand factor, fibrinogen, fibronectin, Factor II, Factor IIa, Factor V, Factor Va, Factor VIII, Factor VIIIa, Factor IX, Factor IXa, Factor X, Factor Xa, Factor XI, Factor XIa, Factor XII, Factor XIIa, Factor XIII, Factor XIIIa, collagen, vitronectin, laminin, osteopontin, fibrillin, chondroitin sulfate, heparin sulfate, and active fragments thereof.

4 The method of claim **3**, wherein said first ligand is von Willebrand factor or an active fragment thereof.

5 The method of claim **1**, wherein said second ligand is a member selected from the group consisting of von Willebrand factor, fibrinogen, fibronectin, Factor II, Factor IIa, Factor V, Factor Va, Factor VIII, Factor VIIIa, Factor IX, Factor IXa, Factor X, Factor Xa, Factor XI, Factor XIa, Factor XII, Factor XIIa, Factor XIII, Factor XIIIa, collagen, vitronectin, laminin, osteopontin, fibrillin, chondroitin sulfate, heparin sulfate, and active fragments thereof.

6 The method of claim **5**, wherein said second ligand is von Willebrand factor or an active fragment thereof.

7 The method of claim **1**, wherein said first ligand and said second ligand are the same.

8 The method of claim **7**, wherein said first ligand and said second ligand are each von Willebrand factor or an active fragment thereof.

9 The method of claim **1**, wherein said first ligand and said second ligand are different from one another.

10. The method of claim **9**, wherein one of said first ligand or said second ligand is von Willebrand factor or an active fragment thereof.

11. The method of claim **1**, wherein said contacting occurs in the presence of a rotating magnetic field.

12. The method of claim **11**, wherein said rotating magnetic field is rotating at a frequency of from 2000–2500 rpm.

13. A platelet/leukocyte interaction assay method, comprising:

contacting a whole blood or blood-derived sample containing platelets and leukocytes with a platelet/leukocyte interaction assay reagent in the presence of an oscillating or rotating magnetic field, said platelet/leukocyte interaction assay reagent comprising a mixture of magnetic and non-magnetic particles, wherein either said magnetic particles or said non-magnetic particles has bound to an outer surface thereof an amount of a first ligand having an affinity for direct interaction with blood platelets and the other of said magnetic particles or said non-magnetic particles has bound to an outer surface thereof an amount of a second ligand having an affinity for direct interaction with leukocytes; and

detecting the presence or absence of platelet/leukocyte interaction function, a level of platelet/leukocyte interaction, or both, in the whole blood or blood-derived sample containing plateets and leukocytes

14. The method of claim **13**, wherein said sample is whole blood.

15. The method of claim **13**, wherein said first ligand is a member selected from the group consisting of von Willebrand factor, fibrinogen, fibronectin, Factor II, Factor IIa, Factor V, Factor Va, Factor VIII, Factor VIIIa, Factor IX, Factor IXa, Factor X, Factor Xa, Factor XI, Factor XIa, Factor XII, Factor XIIa, Factor XIII, Factor XIIIa, collagen, vitronectin, laminin, osteopontin, fibrillin, chondroitin sulfate, heparin sulfate, and active fragments thereof.

16. The method of claim **15**, wherein said first ligand is von Willebrand factor or an active fragment thereof.

17. The method of claim **13**, wherein said second ligand is a member selected from the group consisting of leukocyte selective antibodies, VCAM-1, fibronectin, laminin, ICAM-1, ICAM-2, ICAM-3, collagen osteopontin, vWf, vitronectin, thrombospondin, mucosal addressin cell adhesion molecule 1 (MadCAM-1), P-selectin, L-selectin, and E-selectin.

18. The method of claim **13**, wherein said first ligand is bound to said non-magnetic particles and said second ligand is bound to said magnetic particles.

19. The method of claim **13**, wherein said first ligand is bound to said magnetic particles and said second ligand is bound to said non-magnetic particles.

20. The method of claim **19**, wherein said platelet/leukocyte interaction reagent further comprises a leukocyte marker compound.

21. The method of claim **20**, wherein said leukocyte marker compound is a fluorescent marker compound.

22. The method of claim **18**, wherein said detecting is performed by monitoring movement of said magnetic particles in response to said oscillating or rotating magnetic field.

23. The method of claim **19**, wherein said detecting is performed by monitoring movement of said magnetic particles in response to said oscillating or rotating magnetic field.

24. The method of claim **20**, wherein said detecting is performed by detection or quantitation, or both, of differences in concentration of said leukocyte marker compound in said sample after coagulation of said sample.

25. The method of claim **13**, wherein said contacting occurs in the presence of a rotating magnetic field.

26. The method of claim **25**, wherein said rotating magnetic field is rotating at a frequency of from 2000–2500 rpm.

* * * * *